THE BACTERIAL CONTENT OF THE PROSTATE AND ITS RELATION TO PROSTATIC ADENOMA

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We have at the present time no adequate explanation of the etiology of tumors, benign or malignant. Irritation, both bacterial and traumatic, is considered by many as an exciting factor, and it was thought interesting to carry out an investigation of the relation of infection to prostatic adenoma.

In this series all the cases selected were those showing prostatic adenoma with the exception of Case 9, which proved to be a prostatic abscess and was included because of its bacteriologic interest.

The only reference found in the literature regarding the rôle bacteria play in prostatic adenoma as obtained from the gland at the operating table is the experiments of Dudgeon and Wallace.[1] They examined the urine, and the prostatic secretion from the gland, and the gland itself, and subjected these to a bacteriologic examination. Their method consisted in obtaining the tissue as soon as the gland was removed, the surface of each specimen was seared with a flat knife, and cultures were made from the interior on blood agar. The prostatic fluid was treated in like manner, and in some cases anaerobic cultures were made. They found the following organisms in their first 14 cases:

Prostate Gland	Cases	Urine	Cases
Sterile	3	Sterile	2
B. coli	2	B. coli	6
B. coli and diplococcus	1	Staphylococcus albus	5
B. coli and streptococcus	1	A diphtheroid bacillus	1
Staphylococcus albus	5		
Staphylococcus albus and streptococcus	1		
Staphylococcus albus and citreus	1		

In another series of 14 cases[2] they made bacteriologic examination of the prostate gland only and found the following:

	Cases
Sterile	4
Staphylococcus albus	3
Staphylococcus albus and other cocci	4
B. coli and a diplococcus	1
A new pathogenic bacillus	1
Streptococcus and B. proteus vulgaris	1

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[1] Brit. Med. Jour., 1904, 2, p. 1744.

[2] Wallace: Prostatic Enlargement, p. 83.

They recovered 28 organisms from 21 of the 28 cases (75%). Staphylococcus albus was the commonest organism isolated, and the next most frequent was B. coli, while the gonococcus was not once isolated. This is not at all surprising in view of the difficulty of obtaining a suitable medium for the growth of the gonococcus. A point of extreme interest is that in their first series of the 14 cases in which they recovered organisms from both the prostate and the urine, they were always the same, but in some cases an organism was recovered from the gland and none from the urine and vice versa. They conclude that:

Micro-organisms cause a certain amount of inflammation which produces enlargement of the gland, but it is a secondary event, and similar to that which occurs in tumors elsewhere in the body.

That a bacteriologic examination of the urine may throw little or no light in a similar examination of the prostate.

There is no evidence to support the view that enlargement of the gland is of a gonorrheal origin.

Though we attacked the problem with the newer bacteriologic methods, the results were essentially the same.

As this study was undertaken primarily to investigate the etiology of prostatic hypertrophy it is interesting to follow the cycle of the various theories concerning this subject.

DeSault[3] was the first to suggest the possibility of prostatic hypertrophy being due to an inflammatory process, basing this view on the observation that it was common to those who had had numerous attacks of gonorrhea. Home,[4] quoted by Lydston,[5] suggested the mode of living ("high liver") as one of the predisposing causes. Mercier[6] believes a sluggish circulation as the factor, while Astley Cooper[7] says it is a result of old age and is a physiologic condition, this view being also shared by Squier.[8] Guyon,[9] Launios,[10] and Regnauld[11] believe that it is a senile change, that is, a local manifestation of a general arterio-sclerosis, a fibrosis incident to advancing years, while Casper[12] and Motz[13] have shown that prostatic hypertrophy can and does exist without sclerosis. Wilson, mentioned by Churchman,[14] cites celibacy on the one hand

[3] Oeuvres Clin., 1813, 3, p. 238.
[4] Treatment of the Diseases of the Prostate, 1818, quoted by Lydston.[24]
[5] Etiology of Prostatic Hypertrophy, 1893.
[6] Rech. Anat. Surg. les Mal. d. Org.-Urin. et Gen., 1841, p. 218.
[7] Lectures on the Genitourinary Organs, 1824.
[8] Med. News, 1901, 78.
[9] Ann. d. mal. d. org. genito-urin., 1893, p. 101.
[10] De l'appareil urinaire de viellards, Thèse, 1885, p. 51.
[11] Jour. de l'anat., Par., 1892, 28, p. 109.
[12] Genito-urinary Diseases.
[13] XIII congr. internat. de med., 1900.
[14] Maryland Med. Jour., 1904, 47, p. 400.

and venereal excesses on the other as the probable causes. Amoussat, also quoted by Churchman, considers syphilis as a predisposing factor, and a unique belief is that of Lydston[15] and Civial quoted by Churchman[14] that calculus and stricture may be true causes. A puzzling statement is that of Cabot and Young, Jr.,[16] quoting Cabot and Smith, saying, "that the presence of a previous inflamatory prostatitis prevents later hypertrophy; a stricture and hypertrophy are never present together." Case 31 is in direct opposition to this statement.

Velpeau[17] and the French school consider the enlargement as a neoplastic one; the cause is as unexplained as that of tumors elsewhere in the body, and may be due to an irritation which may itself be due to micro-organisms. This view is the one most adhered to at the present time. Harrison[18] and Daniel[19] conclude that it is a compensatory change in which the bladder is first involved. An extremely interesting belief is that of White,[20] shared by Moullin[21] and Harrison[22] who believes there is a relation between the testes and the prostate, based on first, sexual congress, and secondly on the atrophy of the normal prostate after castration in animals (except dogs) and youths. They advocated the therapeutic application of this theory, that is, to castrate to prevent hypertrophy. To offset this theory Moses[23] cites a case of prostatic hypertrophy in a man 68 years old several years after a complete castration, showing that prostatic hypertrophy is independent of the presence or the absence of the testes.

In his admirable work, Ciechanowski,[24] showed that changes occurred simultaneously in glandular and stromal portions of the prostate gland and that inflammation is the direct cause (and that this was gonorrhea), as was brought out by his pathologic studies, demonstrating the presence of small round cell infiltration. The glandular change was due to a dilatation of the glandular tubules and not to the formation of new ones. In support of this view are Herring,[25] Gouley,[26] Dainiel, Barnette,[27] Finger,[28] and others. Green and Brooks[29] in 1902 publish their results. They selected 58 cases for histologic studies, 6 of them were obtained from operations and the rest from necropsies, and they conclude that the inflammatory theory is the true one, and that true neoplasms of the prostates are rare. Two years later Rothschild[30] had examined 30 prostates removed from the cadavers of patients who had died between the ages of 34 and 52 without evidence of history of disease of the genito-urinary tract. In 27 he found changes similar to those

[15] Am. Jour. Urol., 1908, 4, p. 453.
[16] Ref. Handbook Med. Sc., 1907-8, 7, p. 323.
[17] Leçons orales de clinic de chir., 1841, 3, p. 478.
[18] Brit. Med. Jour., 1895, 2, p. 1605; ibid., 1886, 2, p. 438; Lancet, 1899, 2, p. 126.
[19] Ibid., 1904, 2, p. 1140.
[20] Ibid., 1894, 1, p. 1353.
[21] Lancet, 1896, 1, p. 288.
[22] Ibid., 1900, 2, p. 96.
[23] Therap. Monatsh., December, 1895.
[24] Anat. Intersuch. u. die 590, "Prostate Hypertrophie" u. verus proz. M III. aus der med. u. chir., 1900, 5, p. 183; Ann. d. mal. d. org. gen.-urin., 1901, 19, p. 523.
[25] Brit. Med. Jour., 1904, 2, p. 1136.
[26] Jour. Cutan. and Gen.-Urin. Dis., 1898.
[27] Med. News, 1904, 85, p. 863.
[28] Wien. med. Wchnschr., 1890, 182, 200, 224, 263.
[29] Jour. Am. Med. Assn., 1902, 38, p. 1051.
[30] Centralbl. f. d. Krankh. d. Harn u. Sex. Org., 1904, 15, p. 177.

demonstrated by Ciechanowski and Finger in gonorrheal prostatitis, and he infers that the anlage of prostatic hypertrophy is laid down years before the gland actually begins to enlarge and that gonorrhea is at least the usual cause. Grandon,[31] in his splendid review of the literature of prostatic hypertrophy concludes that the process is a slow formation of connective tissue due to infection, chiefly gonorrheal, as it is the most common, and excludes all other inflammatory causes because of their rarity.

Lydston[15] gives as the cause of prostatic hypertrophy:

1. Inflammation—infectious or traumatic.
2. Chronic prostatic hyperemia and sexual irregularities and excesses.
3. Transpelvic infection by the colon bacillus.
4. Calculi.
5. Senile prostatitis.

Dudgeon and Wallace[1] inclined toward the inflammatory theory, but later Wallace[2] accepted the neoplastic theory as the true one. Rovsing[32] believes that there is first a hypertrophy resulting from hyperemia and that in this chronic process there is destruction of the epithelium and the formation of connective tissue resulting in a shrinkage of the gland, and that the hypertrophy is a compensation, while the qualitative deterioration of the secretion is compensated by the quantitative increase. He showed that of 140 patients with the symptoms of prostatic hypertrophy 40 gave a history of gonorrhea.

From a clinical standpoint Keys,[33] Cunningham and Watson,[34] Churchman, Casper and Legneu[35] are opposed to the inflammatory theory. Posner[36] concludes from his studies that the fat, that is, the lecithin, exerts a chemotactic irritation on the white blood corpuscles, and he claims a relationship between chronic prostatitis and hypertrophy, but believes that hypertrophy is primary and that the inflammatory change is secondary to it, even without infection. Goldberg[37] expresses a similar view. He found the secretion of a patient with prostatic hypertrophy who never had gonorrhea and had never been catheterized, to contain pus cells in the secretion which was scanty and difficult to obtain.

These views, so conflicting in character, apparently are difficult to conciliate; and yet it is considered possible that not any of these theories is the correct one, but that a combination of several may be the exciting causes.

In the early part of my experiments for the purpose of studying the bacterial content of the prostate I devised a method for surface sterilization and concluded that 10 seconds in paraffin oil at 180 C. would be sufficient to destroy surface organisms and permit one to draw definite conclusions from glandular bacterial studies. In those

[31] Ann. Surg., 1902, 36, p. 813.
[32] Archiv. f. klin. Chir., 1904, 74, Part 4.
[33] Jour. Am. Med. Assn., 1904, 43, p. 187.
[34] Watson and Cunningham.
[35] Traite Surg. d'urol.
[36] Am. Jour. Urol., 1904, I, p. 215.
[37] Folio Urologica, 1907.

experiments, the staphylococcus was used as a control on the method employed, but later, using B. coli as a control, growth was obtained; that is, 10 seconds at 180 C. in paraffin oil was not sufficient to kill B. coli. The thermal deathpoint of the colon bacillus was later determined and found to be 20 seconds. Knowing that B. coli is thermostabile, the 20 seconds was accepted as the length of time required to sterilize the surface of glandular tissue, as is seen in Table 1.

As the field of operation is in close proximity to the rectum, and to secure pieces of tissue from the living body (especially this region) without contamination of the surface of these pieces is beyond doubt extraordinarily difficult, we feel that 20 seconds should be used, for though one undoubtedly runs a risk of destroying thermolabile organisms within the tissue, yet if a less effective method is used it is impossible to be certain that surface contamination has not influenced the results, as can easily be seen from the cases in which a control was made to determine that the time of surface sterilization was sufficient to destroy all the surface organisms. I did not begin this until the 31st case, at which time I had proven the technic to be effective. The results up to that time can only be of small interest and any conclusion drawn must be accepted with much reserve.

TECHNIC

A sterile glass jar containing an evaporating dish, scissors and forceps are kept ready, and as soon as the operator enucleates a prostatic lobe, the lid is removed from the jar and the tissue is dropped by the operator or the assistant into the jar. This is carried to the laboratory and emulsified in the sterile air chamber as follows:

Hands are scrubbed, washed and soaked in bichlorid solution, introduced into the gloves of the sterile air chamber, the mortar is put underneath the opening, into which 25 c c of dextrose ascitic broth is poured. The flange is flamed and closed; the oil, which is in a tall narrow pyrex beaker to prevent ebullition, is heated up to 185 C. and allowed to come down to 180 C.; the tissue is immersed in the oil for 20 seconds after which it (tissue and mouse teeth clamp) is dropped into the mortar after flaming the flange, there to be emulsified and poured into a large test tube. To check this method, a piece of autoclaved kidney was dipped in a 24-hour broth culture of staphylococci (later B. coli was used), heated, as the prostatic tissue was, and run through a similar process. The emulsions were then removed from the chamber and anaerobic and aerobic cultures were made, using hydrocele or ascitic fluid, and the emulsions were then put in the refrigerator for future use. Pieces of prostatic tissue were transferred from the emulsion to the culture tubes, and sealed with paraffin to facilitate anaerobic conditions, and kept in the incubator for at least 3 weeks before discarding. One c c of the emulsion was plated at once. The time of appearance and general character of the colonies, their distribution and subcultures made on various

media, both aerobically and anaerobically, were noted. In most cases the pathologic report as made by the resident pathologist and their interpretation are given.

Beginning with the twelfth case it was considered advisable to run a control to note the organisms contaminating the surface as received directly from the operator. As soon as the tissue was brought to the laboratory 10 c c of broth was poured over the gland and allowed to remain 5-10 minutes, 1 c c of this being then plated. From here on the technic was the same as before. In Table 2 a complete analysis of the cases is tabulated.

TABLE 1

PRELIMINARY EXPERIMENTS ON STERILIZATION OF SURFACE OF TISSUE AFTER INOCULATION

Length of Time Tissue Was Heated in Oil at 180 C.							
10 Sec.	12 Sec.	15 Sec.	18 Sec.	20 Sec.	22 Sec.	25 Sec.	30 Sec.
+	+	+	+	+	0	0	0
+	+	+	+	+	0	0	0
+	+	+	+	0	0	0	0
+		0	0	+	0	0	0
+		0	0	0	0	0	0
+		0	0	0	0	0	
+		0		0	0	0	
+		0		0		0	
+		0		0		0	
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		+					
		+					
		0					

Explanation: + = growth; 0 = sterile plate.

DISCUSSION

From Table 1 it is seen that the thermal deathpoint of B. coli lies between 15 and 20 seconds in paraffin oil at 180 C. We also see why in several of the control experiments, 15 seconds was sufficient to kill B. coli in Cases 27 and 28, for we find a similar condition in these experiments. Again we see that 10 seconds seemed to be sufficient to kill B. coli in 6 series. For these divergent results no definite explanation can be offered other than that the organism may have been of a different strain; of lower resistance; fewer in number or that the size of the tissue used may have varied; or a combination of

these factors may account for the results. It is certain that 25 seconds will not kill some organisms that may be on the interior of the gland, as can be seen in Cases 31, 32 and 33 from which staphylococci were isolated. I selected 20 seconds as the time to destroy B. coli on the exterior. When this time was employed 3 showed positive growth out of 18 cases.

From this series of 39 cases it is seen in Tables 2 and 3 that 14 organisms have been isolated (36%). More organisms were recovered but they cannot be included in the group, that is, as coming from the prostatic tissue proper, because the technic was not sufficiently developed until the 31st case. I did include those cases in which the plate poured immediately after the emulsion was made showed innumerable colonies; also where there was a co-existing abscess, and in which the surface organism was of one type while that isolated from the emulsions was of another kind.

The fact that more bacteria were not isolated may be explained by the fact that the organism either had died out in the tissue or had become attenuated and thermolabile and the only signs of inflammation were the small round cell infiltration.

As regards the relationship between prostatic hypertrophy and gonococcus infection, Table 2 shows that of the 39 cases in this series 29 patients (74.7%) denied infection, while only 10 admitted (20.3%), so that the anamnesis shows that individuals with a negative history are as susceptible, if not more so, to prostatic hypertrophy, as those who have had a gonococcus infection. In the 4 patients from whom an organism was isolated not one admitted having gonorrhea.

The micro-organisms found on the surface of the gland, in 29 of the cases, were for the most part similar to those isolated from the prostatic emulsion. In several cases, however, Cases 18, 31, 32, 39 and 40, the organisms were different. In Cases 13, 21 and 24 no surface organisms were found. In the last case it is peculiarly interesting to note that while the surface culture did not yield an organism, the prostatic emulsion yielded a facultative anaerobic gram-positive staphylococcus, so that the surface organism need not in all cases be considered as contaminators; for, in some cases, the bacteria may be in the prostate gland and may be exposed by the cutting of the tissue. In urine no attempt was made to determine the type of organism present as done by Wallace and Dudgeon, other than whether it was a coccus or a bacillus.

TABLE 2

RESULTS OF CULTURES

Case	Gonorrhea	Urinalysis	Method of Surface Sterilization		Organisms Isolated	Surface Organisms		Control on Method			Pathologic Diagnosis	Remarks
			Medium	Time		No.	Type	Time	Org.	Result		
1	Denied	Coccus and bacillus	Salt solution	15 sec.	G.* Streptococcus? Streptobacillus?	.....	.....	....	....	....	1. Round cell infiltration. 2. Benign hypertrophy, fibroglandular. 3. Chronic prostatitis.	Method of sterilization is questionable here.
2	Denied	Coccus and bacillus	Flame. Water not Sterilized	15 sec.	0 B. coli? B. coli	.....	.....	....	....	....	1. Round cell infiltration. 2. Benign hypertrophy, fibroglandular.	Contamination?
3	Denied	Bacillus	Flame	Passed through flame several times	B. coli	.....	.....	....	....	....	Marked peri-acinus small round cell infiltration. P. H., F. O.; chr. prostat.	This organism we believe to have been on the interior of the gland.
4	Denied	Coccus and bacillus	Flame, water	8 times, 20 sec.	0	.....	.....	....	....	....	No round cell infiltration. P. H., F. G.	No organisms and no round cell infiltration.
5	Denied	0	Oil	5 sec.	0	.....	.....	5 sec.	Staphylococcus	0	.....	Organism from swab of seminal vesicles lost in transferring.
6	30 years ago	Coccus and bacillus	Water	7 sec.	0	.....	.....	7 sec.	Staphylococcus	+	Round cell infiltration. P. H., F. G.; chr. prostatitis.	No organism isolated. Control contaminated, so time is lengthened in next case.
7	Denied	Bacillus	Water	10 sec.	0	.....	.....	10 sec.	Staphylococcus	+	Marked round cell infiltration. P. H., F. G.; chr. and subacute prostatitis.	No organism isolated. Control positive growth.
8	46 years ago	Coccus	Water	10 sec.	0	.....	.....	10 sec.	Staphylococcus	+	Marked round cell infiltration. P. H., F. G.; chr. prostatitis.	No organisms isolated.
9	10 weeks ago	0	....	....	B. coli communis	.....	.....	....	....	....	.....	This case was one of prostatic abscess and B. coli communis was isolated.
10	25 years ago	Bacillus	Paraffin oil	10 sec.	0	.....	.....	10 sec.	Staphylococcus	+	Moderate grade of round cell infiltration. P. H.; chronic prostatitis.	Here an organism should have been isolated, if any relation is to be drawn from the pathological sections unless it was very thermolabile.
11	Denied	Bacillus	P. O. 180 C.	10 sec.	0	.....	.....	10 sec.	Staphylococcus	+	Moderate grade of small round cell infiltration. P. H., F. G.; chr. prostatitis.	Here an organism should have been isolated, if any relation is to be drawn from the pathological sections unless it was very thermolabile.

12	Denied	Coccus and bacillus	P. O. 180 C. Saline solution	6 sec. 20 sec.	B. coli?	2,530	1. B. coli 2. Staphylococcus albus 3. Streptococcus	6 sec.	Staphylococcus	0	Slight grade of round cell infiltration. P. H., F. G.; chr. prostat.	We cannot accept the B. coli without question in this case, for the surface was well covered with organisms. We only obtained it from the oil method, while flame and 20 sec. in saline was negative.
13	Several times	0	P. O. 180 C.	10 sec.	00	0	0	10 sec.	B. coli	+	Slight grade of small round cell infiltration. P. H., F. G. type.	On account of B. coli in previous case it was used as a check to control method in this case.
14	Denied	Bacillus	P. O. 180 C.	6 sec. 10 sec. 15 sec.	B. coli communior ?	1,500	B. coli communior; Staphylococcus albus; Streptococcus	10 sec.	B. coli	+	Marked round cell infiltration. P. H.; chr. prostatitis	Again we see that 10 sec. was not sufficient to completely sterilize the surface of gland.
15	Denied	0	P. O. 180 C.	8 sec.	B. coli communis	3,000	B. coli communis	8 sec.	Staphylococcus	0	Round cell infiltration. P. H., glandular abscess.	B. coli communis was isolated from interior as well as surface.
16	Denied	Coccus and bacillus	P. O. 180 C.	10 sec.	B. coli communis	.....	B. coli	10 sec.	Staphylococcus	0	Moderate grade of small round cell infiltration. P. H., fibro-cystic type; chronic prostatitis.	B. coli was isolated but cannot be accepted as in gland proper; method was inefficient.
17	Denied	Bacillus	P. O. 180 C.	10 sec.	0	.....	B. coli	10 sec.	Staphylococcus	0	Moderate grade of small round cell infiltration, P. H., glandular; chr. prostatitis.	This case is interesting for 10 sec. seemed to destroy the B. coli and can only explain it on the fact that there were only a few organisms.
18	Denied	Coccus	P. O. 180. C.	10 sec. 15 sec	B. welchii 0	200	Staphylococcus albus	10 sec.	Staphylococcus	0	Slight grade of small round cell infiltration. P. H., F. G. type.	Isolated B. welchii, but believe it to be a contaminator.
19	30 years ago	Coccus and bacilli	P. O. 180 C.	10 sec.	0	100	Staphylococcus albus	10 sec.	Staphylococcus	0	Marked small round cell infiltration.	No organisms isolated.
20	Denied	Bacilli	P. O. 180 C.	10 sec.	Gas producing organism. Anaerobic	5,000	?	10 sec.	Staphylococcus	0	Moderate amount of small round cell infiltration. P. H., F. G.; chr. prostatitis.	Failed to isolate organisms.
21	Denied	Bacillus and cocci	P. O. 180 C.	10 sec.	0	0	.....	10 sec.	Staphylococcus	0	Marked grade of small round cell infiltration. P. H., F. G.; chr. prostatitis.	This is the case in which the surface culture was negative, and the section showed a marked round cell infiltration.
22	Denied	Coccus	P. O. 180 C.	10 sec.	0	550	Staphylococcus aureus and albus	10 sec.	B. coli	+	Moderate grade of small round cell infiltration. P. H., F. G. type.	Control again was ineffective.
23	20 years ago	Bacillus	P. O. 180 C.	10 sec.	Staphylococcus	4,000	Staphylococcus albus	10 sec.	B. coli	+	Slight grade of small round cell infiltration. P. H., F. G. type.	There is no question about the organism being from the gland but the control is again found at odds.

TABLE 2—*Continued*

RESULTS OF CULTURES

Case	Gonorrhea	Urinalysis	Method of Surface Sterilization		Organisms Isolated	Surface Organisms		Control on Method			Pathologic Diagnosis	Remarks
			Medium	Time		No.	Type	Time	Org.	Result		
24	Denied	No record	P. O. 180 C.	10 sec.	Facultative anaerobic staphylococcus; gram-positive	0	0	10 sec.	Staphylococcus	0	Slight grade of small round cell infiltration. P. H.	This is the 3rd case in which the surface culture was negative, isolated gram-positive staphylococcus, facultative anaerobe.
25	Denied	Bacillus	P. O. 180 C.	10 sec.	0	700	B. coli	10 sec.	B. coli	0	Slight grade of small round cell infiltration. P. H., F. G. type.	This case complicated our method by 10 sec. in P. O. at 180 C. which was sufficient to destroy B. coli.
26	Denied	Coccus and bacillus	P. O. 180 C.	10 sec.	B. coli	0	B. coli	10 sec.	B. coli	+	Marked grade of small round cell infiltration. P. H., F. G.; chr. prostatitis.	B. coli was isolated from gland in spite of the positive control.
27	Several	No record	P. O. 180 C.	15 sec.	B. coli?	3,750	B. coli	15 sec.	B. coli	0	Marked grade of small round cell infiltration. P. H., G.; chr. prostatitis.	Though we believe the organism isolated is from the gland proper, we cannot include it as such owing to the surface culture and later work on the T. D. P. B. coli.
28	Denied	Coccus and bacillus	P. O. 180 C.	15 sec.	Bacilli, belonging to the colon group	10,000	?	15 sec.	B. coli	0	Moderate grade of small round cell infiltration. P. H., F.	The organism isolated belonged to the colon group.
29	Denied	Streptococcus; staphylococcus; bacillus	P. O. 180 C.	15 sec.	0	60	Streptothrix	15 sec.	B. coli	+	No section made.	The control showed growth upsetting previous results.
30	At 50	Bacillus	P. O. 180 C.	15 sec.	B. coli communis?	360	B. coli	15 sec.	B. coli	+	Mild grade of small round cell infiltration. P. H., G.	The control was again positive.
31	No history of N	Coccus and bacillus	P. O. 180 C.	25 sec.	B. fecalis alkaligenes	20	B. coli; staphylococcus albus	25 sec.	B. coli	0	No section made.	There is no question about this organism being indigenous to the gland. In this case we find stricture and hypertrophy together.
32	Denied	Coccus	P. O.	25 sec.	Anaerobic bacilli failed to isolate	1,000	1. B. coli. 2. Staphylococcus. 3. Unknown bacilli.	25 sec.	B. coli	0	Moderate grade of small round cell infiltration. P. H., F.	Unable to isolate anaerobic bacillus.

33	Denied	Bacillus	P. O.	25 sec.	Staphylococcus; B. pyocyaneus	10,000	1. B. coli. 2. Staphylococcus. 3. B. pyocyaneus.	25 sec.	B. coli	0	Moderate grade of small round cell infiltration. P. H., G.; prostatic abscess.	There was a prostatic abscess associated with the hypertrophy. A staphylococcus and pyocaneus was isolated showing that 25 sec. will not destroy some organisms on the interior of the gland.
34	Denied	Coccus and bacillus	P. O.	20 sec.	1. Staphylococcus 2. B. Proteus vulgaris?	50	1. B. coli. 2. Staphylococcus	20 sec.	B. coli	0	Marked grade of small round cell infiltration. P. H., F. G. type; chronic prostatitis.	Isolated 2 organisms out of 3, which appeared in surface culture. We are certain the coccus was isolated from the gland, but do not feel the same about the bacillus.
35	Denied	Coccus and bacillus	P. O.	20 sec.	B. coli	5,000	B. coli	20 sec.	B. coli	0	Marked grade of small round cell infiltration. P. H., G. type; chronic prostatitis.	Organism isolated was evidently from the interior of the gland as the control remained sterile.
36	Denied	Bacillus	P. O.	20 sec.	0	200	B. coli	20 sec.	B. coli	0	Moderate grade of small round cell infiltration. P. H., F. G.; chronic prostatitis.	The organism B. coli was on surface of gland.
37	Denied	0	P. O.	20 sec.	0	1,500	Staphylococcus aureus	20 sec.	B. coli	0	Marked grade of small round cell infiltration. P. H., G.; chronic prostatitis.	The reason no organism was isolated may be because it was thermolabile.
38	Denied	Bacillus	P. O.	20 sec.	0	2,000	Staphylococcus aureus	20 sec.	B. coli	0	Marked grade of small round cell infiltration. P. H., F. G.; chr. prostatitis.	As in previous case organism may have been a thermolabile one.
39	Denied	Bacillus	P. O.	20 sec.	B. coli communis. 0 (spore bearer G.)	10,000	Staphylococcus albus	20 sec.	B. coli	0	Slight grade of small round cell infiltration. P. H., G. cystic type.	The surface culture showed staphylococcus and was not obtained from the gland emulsion from which B. coli communis was recovered with a G. + spore-bearing bacillus (contamination).
40	Admitted	Mixed coccus	P. O.	20 sec.	Facültative anaerobe bacillus, G., nonmotile	5,000	B. coli. Staphylococcus albus	20 sec.	B. coli	0	Marked grade of small round cell infiltration. P. H., G.; chronic prostatitis.	The organism was indigenous to the gland and shows 20 sec. will kill B. coli infection.

* P. H.—Prostatic hypertrophy. F.—Fibroid type. G.—Glandular type. F. G.—Fibro-glandular type. P. O.—Paraffin oil.

TABLE 3

ORGANISMS ISOLATED FROM 39 CASES OF THE PROSTATE GLAND

Type of Organism	Found Alone	Associated with Other Organisms	Gonococcus Infection Denied	Gonococcus Infection
B. coli	5		5	
Staphylococcus	1	2	2	
Colon-like bacillus	1		1	
Anaerobic gas forming organism	1		1	
Facultative anaerobe, gram-positive staphylococcus	1		1	
B. fecalis alkaligenes	1		1	
Anaerobic bacilli failed to isolate	1		1	
B. pyocyaneus	0	1?		
B. proteus vulgaris	0	1?		
Unknown bacillus. Facultative anaerobe, gram-negative, G., nonmotile	1		1	

Case 9 was a prostatic abscess and is not included in above table.

TABLE 4

SURFACE ORGANISMS ISOLATED FROM 29 CASES OF THE PROSTATE GLAND

Type of Organism	Found Alone	Associated with Other Organisms
Sterile	3 times	
B. coli communis	9 times	6 times
B. coli communior		1 time
Staphylococcus albus	4 times	8 times
Staphylococcus aureus	2 times	1 time
Streptococcus		2 times
Streptothrix	1 time	
Streptobacillus		1 time
Unknown bacillus		1 time
B. pyocyaneus		1 time
B. proteus vulgarus		1 time
Unknown	2 times	

SUMMARY

Ten seconds is not sufficient to destroy all surface organisms as stated in a previous article.[38]

Twenty seconds in paraffin oil at 180 C. is sufficient to kill B. coli and permit the staphylococcus to be isolated from the interior of the gland, if present, but may destroy more thermolabile organisms.

Organisms were isolated from 14 cases (36%).

The surface and interior organisms were the same in some cases; the former need not necessarily be a contaminator.

There does not seem to be any relationship between the small round cell infiltration, as is seen in the sections of each gland, and the organism isolated. The cellular reaction was present in all but one case in this series.

[38] Davis and Rosen: Jour. Infect. Dis., 1917, 21, p. 323.

The colon group of organisms was the commonest found; no one organism was found specific to the gland; not once was the gonococcus isolated.

The history of the cases in this series shows that persons with a negative record as to gonococcus infection to be as susceptible to prostatic hypertrophy as those with infection.

No comparison can be drawn between the urinary organisms and those isolated from the gland.

The significance of the bacteria isolated from the prostate gland and the rôle that they may play in prostatic hypertrophy, if any (other than that they may be secondary invaders), cannot be determined from this investigation.